COMBINATION ANTHELMINTICS TO CONTROL GASTROINTESTINAL NEMATODES IN FOALS

A Thesis

by

ASHLEY ELIZABETH VOLKER

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

August 2008

Major Subject: Animal Science

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Approved by:

Chair of Committee,	Martha M. Vogelsang
Committee Members,	Josie A. Coverdale
	Thomas M. Craig
Head of Department,	Gary Acuff

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ABSTRACT

Combination Anthelmintics to Control

Gastrointestinal Nematodes in Foals. (August 2008)

Ashley Elizabeth Volker, B.S., University of Vermont

Chair of Advisory Committee: Dr. Martha M. Vogelsang

Two common nematodes that affect young horses are cyathostomes (small strongyles) and *Parascaris equorum* (ascarids). It has been recently found that populations of these nematodes are resistant to common anthelmintics used to control them. Small strongyles have been found to be resistant to pyrantel and fenbendazole, while ascarids have been found to be resistant to ivermectin. This represents a unique dilemma in controlling the gastrointestinal nematode population in the foal. It has been shown in other host species that combination anthelmintics can be used to successfully treat resistant nematodes. The current study utilized 28 foals and was conducted from April to November 2007. The foals were allocated into age cohorts and randomly assigned a treatment regimen. Group I was administered ivermectin at 0.2 mg/kg BW. Group II was administered ivermectin at 0.2-mg/kg BW and pyrantel pamoate at 6.6 mg/kg BW. Group III was administered ivermectin at 0.2-mg/kg BW and fenbendazole at 10 mg/kg BW. Group IV was administered pyrantel pamoate at 6.6 mg/kg BW and fenbendazole at 10 mg/kg BW. Fecal samples were collected at time of treatment and two wk post treatment to determine effectiveness in removing egg producing adult

nematodes. Each age cohort was then treated 30 d later with a different anthelmintic or combination. That is, foals in group I were treated as those in group II, group II to treatment III, group III to treatment IV, and group IV to treatment I. Over a period of 4 mo, each foal received at least one treatment in consecutive order.

The difference of egg counts (pre-treatment minus post-treatment) for small strongyles treated with ivermectin (IVM) was 29.39 eggs per g (EPG), 5.44 EPG for ivermectin with pyrantel (PRT), 3.85 EPG for ivermectin with fenbendazole (FBZ), and -8.32 EPG for pyrantel with fenbendazole. There was a significant difference when comparing IVM to IVM+PRT ($P = 0.0018$), IVM vs. IVM+FBZ ($P = 0.0010$), and IVM vs. PRT+FBZ ($P < 0.0001$). IVM was more effective than each of the other treatments. There was no influence of treatment on ascarid EPG ($P > 0.1184$).

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CHAPTER I

INTRODUCTION

Two gastrointestinal nematodes are of particular importance in foal health. Normally only foals are infected with *Parascaris equorum* (ascarid), and as the young horse is exposed to ascarids, they usually acquire immunity. Thus, the parasite is not typically found in adult horses. Strongyle type worms (40 species in North America) infect all ages of horses, but the unique combination of cyathostomes (small strongyles) and ascarids is only found in foals. Foals do not exhibit patent infections of either parasite until they are approximately 2 mo of age, due to the long prepatent period, the time from infection until mature, egg-laying adults develop.

Historically anthelmintics, particularly ivermectin, have been used to control these nematodes in foals. Ivermectin continues to be extremely effective against cyathostomes, but may not be as effective against ascarids (Boersema et al., 2002; Hearn and Peregrine, 2003; Lyons et al., 2006; Slocombe et al., 2007; Craig et al., 2007; Lindren et al., 2008). Other anthelmintics, pyrantel and fenbendazole, are effective against ascarids but have a diminishing effect on the small strongyles (Lyons et al., 1996; Lyons et al., 1999; Tarigo-Martinie et al., 2001; Kaplan, 2002; Kaplan et al., 2004). This represents a unique problem, one parasite is resistant to one class of anthelmintic and another parasite is resistant to another class, all while being in the

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This thesis follows the style and format of the Journal of Animal Science.

same host. In other host species, such as sheep and goats, the use of anthelmintic combinations has been tested with positive results against nematodes resistant to multiple classes of anthelmintics (Miller and Craig, 1996). The objective of this study was to determine whether combinations of anthelmintics can be utilized to effectively control the gastrointestinal nematodes, small strongyles and *Parascaris equorum*, in foals.

CHAPTER II

REVIEW OF LITERATURE

Parascaris equorum

The first, *Parascaris equorum* (ascarids) may cause coughing and nasal discharge, due to larval migration from the intestines through the lungs, reduced weight gain, lack of condition, lethargy, depression, and even death from intestinal obstruction of mature ascarids (Clayton and Duncan, 1978; Clayton, 1986). Infections of ascarids are acquired naturally, soon after birth by ingestion of infective eggs during grazing, or investigation of their surroundings if housed indoors. Ascarid eggs are passed in feces and may become infective within 10 d under suitable environmental conditions. If conditions are favorable, eggs can remain viable for years due to the thick, three-layered protective shell, which provides environmental resistance. The mature larvated egg hatches in the intestinal tract after ingestion by a foal. Within 24 h, larvae penetrate the liver and spend 1 wk traveling through the hepatic parenchyma before moving on to the lungs. Larvae are present in the pulmonary parenchyma and airways for an additional week. Larvae return to mature in the small intestine via the tracheoesophageal junction and can be found in the duodenum as early as 2 wk after infection. The prepatent period, time from infection to beginning of egg production by female adults, is variable, with a range of 72 to 110 d. Adult ascarids are extremely fecund, with an approximate output of greater than 50 million eggs shed from a foal per day (Clayton, 1986).

Parascaris is a highly antigenic and immunogenic parasite of foals. Foals develop acquired immunity to the worm by the time they are one year of age. Bello et al. (1974) found that as precipitin titers to whole-worm *Parascaris* antigen increased, the number of eggs per gram in fecal material decreased. Titers increased gradually from a nonspecific reaction at birth to a titer of 24 by 5 to 8 wk, maintained these levels until wk 20, and then rose sharply to wk 28. The titers continued to increase as yearlings developed an active protective immunity (Bello et al., 1974 and Bello, 1985).

Small strongyles

The second group of gastrointestinal nematodes important to foal health are small strongyles (cyathostomes). There are over 40 species in North America belonging to the nematode subfamily (*Cyanthostominae*). The eggs produced are similar to those of large strongyles (*Strongylus vulgaris*) but cyathostomes usually comprise over 95% of the strongylid eggs in a fecal sample. Small strongyles infect all ages of horses, but the unique combination with ascarids is only found in young horses (Reinemeyer, 1986).

Clinical signs of naturally acquired cyanthostome infections include loss of weight and decreased rate of gain, emaciation, general debility, and possible progression to mortality. Experimental infections have resulted in pyrexia, tachycardia, anorexia, diarrheic stools, listlessness, and failure to shed winter hair coat. Experimental infections have also resulted in decreased or changes in enterocolic motility, which is of great importance, since changes in motility are commonly associated with colic in horses (Reinemeyer, 1986). Larval cyanthostomiasis is common in young horses (1 to 3 years of age), and is related to the emergence of excysted larvae from the walls of the large

intestine or penetration of the mucosa by infective 3rd stage larvae (L_3) strongyles. Clinical signs include weight loss, diarrhea, pyrexia, and subcutaneous (especially ventral) edema (Lyons et al., 2000).

Horses become infected with cyathostomes by ingestion of the L_3 larva during grazing. They have a five-stage direct life cycle with adult females living in the cecum and large intestine of the horse and producing eggs that are passed in the feces. Under favorable environmental conditions (temperature and moisture being the most crucial), an embryo develops within the egg and the first stage larva hatches. Further development occurs to the second and third stage larvae. The infective L_3, encased in a sheath, resides on vegetation where it is then ingested by the grazing horse. Infective L_3 larvae are exsheathed in the small intestine and penetrate the mucosa in the cecum and large intestine. They molt to fourth stage larvae (L_4) within a tissue cyst of host origin within 6 to 12 d. They arrest within the cysts for 1 to 2 mo before emergence and entrance into the lumen of the large bowel and develop into adults. Emergence is often seasonal, occurring in the first month of the yr in a northern temperate climate. The prepatent period varies among species of parasite and age of host, usually between 5.5 to 14 wks. Adult cyathostomes are plug feeders, removing small bits of large intestinal mucosa as they feed, resulting in mucosal ulceration (Reinemeyer, 1986 and Lyons et al., 2000).

Anthelmintics

In order to control the nematodes within the young horse, anthelmintics (antiparasitic drugs) are administered along with management practices to control

exposure. When using anthelmintics, the intention is to remove the adult egg-laying stages from the gastrointestinal tract of the animal, and therefore lessen the effects on the animal and environmental contamination (Drudge and Lyons, 1966). There are three major classes of anthelmintics currently used to control gastrointestinal nematodes in horses: benzimidazoles (fenbendazole, oxfendazole, oxibendazole, others), tetrahydropyrimidines (pyrantel salts), and avermectin/milbemycins (macrocylic lactones; ivermectin and moxidectin). Piperazine and phenothiazine have been used in the past, but are presently infrequently used. When first introduced, all of these drugs had good to excellent efficacy against cyathostomes and ascarids. However, it is an increasing concern that both nematodes are developing resistances to one or more of these drugs (Kaplan, 2002).

When first introduced in the 1980's, ivermectin (22,23-Dihydroavermectin B_1) was extremely effective against small strongyles and ascarids in young and mature horses. In early studies, there was a >99% reduction in the number of small strongyles when ivermectin was administered via injection or oral paste at a rate of 0.2 mg ivermectin/kg BW (Klei and Torbert, 1980; Craig and Kunde, 1981; Yazwinski et al., 1982a). Similarly, there was a 96 to 100% reduction in adult *Parascaris* (Egerton et al., 1981; Yazwinski et al., 1982b) and a 98.5% reduction in immature ascarids when administered IM at 0.2-0.3 mg/kg BW (Yazwinski et al., 1982b). Ivermectin acts on the nematode by enhancing the effect of the neurotransmitter γ-aminobutyric acid on the nervous system, causing irreversible, flaccid paralysis. The nematode can then no longer maintain its position in the digestive tract and is eliminated (Austin et al., 1991).

The second class of anthelmintics, benzimidazoles, exerts antiparasitic activity by inhibiting nematode energy metabolism. Starvation of the worm results from inhibition of microtubular polymerization, decreasing nutrient absorption by the tegmental and intestinal cells (Austin et al., 1991). DiPietro et al. (1984) found that 10 mg/kg BW of fenbendazole (a benzimidazole) was highly effective against both immature and mature *Parascaris* when experimentally infected. Similarly, it was found that fenbendazole given for 5 d at a rate of 10 mg/kg BW killed the migrating larvae (DiPietro, 1984). When given at a rate of 7.5 mg/kg BW for 5 d consecutively, fenbendazole resulted in a 90% reduction in adult cyathostomes and an approximately 95% reduction in the larval stages (Duncan et al., 1998).

The third class, tetrahydropyrimidines or pyrantel salts, are acetylcholine agonists and cause continuous firing of impulses at the neuro-muscular junction of nematodes, resulting in tonic paralysis (Austin et al., 1991). Pyrantel pamoate (trans-1-methyl-1,4,5,6-tetrahydro-2[2 (2 thienyl)vinyl] pyrimidine pamoate) was found to have a 92 to 100% efficacy against adult ascarids and a 100% efficacy against immature ascarids when fed at a rate of 6.6 mg/kg BW via stomach tube or with feed (Lyons et al., 1974). Pyrantel pamoate also had an 89 to 96% efficacy against small strongyles (Lyons et al., 1974). In contrast to these early reports the effectiveness of anthelmintics, nematodes have developed resistance to one or more class of drug(s).

Resistance

Clayton (1980) first defined resistance as a fecal egg count reduction of less than 70% at 7 to 14 d post treatment. However, this is probably somewhat of a conservative

measure. The World Association for the Advancement of Veterinary Parasitology (WAAVP) published recommendations for standardizing procedures to detect nematode resistance (Coles et al., 1992). They defined resistance in sheep and goats as a reduction in fecal egg counts of less than 95% with a lower confidence limit of less than 90%. There was only a brief mention of the problem in horses, indicating a reduction in fecal egg counts of less than 90% as indicative of benzimidazole resistance. This is the only drug class they mentioned and gave no justification for this recommendation (Bauer et al., 1986). Kaplan (2002) advised that there must be established standards for performing and analyzing fecal egg count reduction tests in order to evaluate the magnitude and prevalence of resistance internationally. Furthermore, since there are variations in efficacies among drug classes against susceptible cyathostomes, a universal standard is probably not useful. The previously recommended 90% reduction level is probably fair for benzimidazoles for declaring resistance. However, ivermectin treatment consistently gives nearly a 100% reduction in FEC, therefore the appearance of any eggs following treatment is cause for concern. Accordingly, a definition of resistance for ivermectin of < 95% may be too conservative, and a more stringent definition is required. Pyrantel has variable and much lower efficacies, even when first introduced; consequently a much more conservative definition of resistance is required (Kaplan, 2002).

The first report of small strongyle drug resistance in horses was to phenothiazine, which is no longer available in the United States. In the mid-1960's, small strongyles were found to be resistant to thiabendazole (Lyons et al., 1999). Drudge et al. (1979)

found five species of small strongyles to be resistant to thiabendazole, along with resistance to mebendazole, cambendazole, fenbendazole, and oxfenbendazole with fecal egg count reductions of only 40 to 98%. Only oxibendazole was 100% effective with a complete elimination of small strongyles (Drudge et al., 1979). Widespread benzimidazoles-resistant small strongyles are found in the United States (Lyons et al., 1996; Lyons et al., 1999; Tarigo-Martinie et al., 2001; Kaplan, 2002; Kaplan et al., 2004) and 20 other countries (Lyons et al., 1999).

Small strongyle resistance to pyrantel has also been reported in the United States (Chapman et al., 1996; Woods et al., 1998; Lyons et al., 2001; Tarigo-Martinie et al., 2001), Norway (Ihler, 1995) and Denmark (Craven et al., 1998; Craven et al., 1999). It is speculated that the common practice of low-dose (2.64 mg/kg BW) daily feeding of pyrantel tartrate is one of the primary factors for increasing resistance in the United States (Kaplan, 2002). The study conducted by Tarigo-Martinie et al. (2001), found the mean reduction of egg counts to be only 89% at 14 d post treatment with pyrantel on 7 farms. One farm had no reduction in fecal egg count (FEC) 2 wk after treatment, indicating that resistance of cyathostomes to pyrantel was high on that farm. This was the only farm with a history of daily pyrantel tartrate use, giving support to the theory of this facilitation of resistance (Tarigo-Martinie et al., 2001).

The first observation of an equine nematode showing resistance to ivermectin was found in the Netherlands in 2000. Boersema et al. (2002) found that ivermectin was ineffective in controlling *Parascaris* infection in a small number of foals on a single farm. Since then *Parascaris* resistance to ivermectin has been identified in Canada

(Hearn and Peregrine, 2003; Slocombe et al., 2007), Kentucky (Lyons et al., 2006), Texas (Craig et al., 2007), and recently Sweden (Lindgren et al., 2008). Hearn and Peregrine (2003) observed a high number of ascarid eggs 12 to 13 d after treatment with ivermectin. In Canada, it was found that the overall efficacy of ivermectin was only 34% in foals, while fenbendazole and pyrantel pamoate had an efficacy of 98% (Slocombe et al., 2007). On the Texas horse farm, the same one as used in the present study, it was found that ivermectin was inadequate in removing ascarids after treatment, and there was even a significant increase in egg counts indicating maturation of worms already present in the intestine. The product, however, was more effective in foals greater than 8 mo of age (Craig et al., 2007).

This represents a unique problem in the young horse whereas they are plagued by two different nematodes that are resistant to different anthelmintics. One nematode (small strongyles) is resistant to more than one type of drug (pyrantel and fenbendazole), whereas the other nematode (*Parascaris*) is resistant to a different class (ivermectin). Therefore, an adequate method of control for both must be found. Considerations when administering treatments against resistant nematodes include: realization of the additional cost of increasing dosages of anthelmintics and possible toxic reactions to the drugs. The toxicosis factor for fenbendazole is 100x the recommended dose; it is 20x the recommended dose for pyrantel; but only 6x the recommended dose for ivermectin. Thus care must be taken as to not cause adverse effects in young horses (Wescott, 1986).

Anthelmintic combinations

In other host species, such as sheep and goats, combinations of anthelmintics have been used with positive results against resistant nematodes (Bennet et al., 1980; Anderson et al., 1988; Waller et al., 1990; Anderson et al., 1991; Miller and Craig, 1996). Combinations do not produce toxic effects in the host because of differing modes of action and the administration of each drug at its recommended dose. Resistance by sheep and goat nematodes, such as *Haemonchus contortus*, to more than one class of anthelmintic is highly prevalent. It is debated as to whether the combinations of drugs act by a synergistic or an additive effect. Bennet et al. (1980) described a synergistic effect, as the two drugs used had different modes of action; therefore a possible interaction was obtained. They found that treatment with mebendazole or levamisole alone caused no significant decrease in egg counts when administered to sheep with benzimidazole resistant *Haemonchus contortus*. However, when administered together, a reduction of 80% was found (Bennet et al., 1980). Anderson et al. (1991) described an additive effect, although comparison of the two studies is difficult because different worms and drugs were investigated. There was a reduction in resistant *Ostertagia* species and *Trichostrongylus colubriformis* of 97 to 98% and 95 to 100%, respectively when sheep were administered combinations of albendazole and levamisole. Reductions of only 80 to 90% (*Ostertagia*) and 36 to 86% (*T. colubriformis*) were found when the drugs were administered singly (Anderson et al., 1991). Miller and Craig (1996) found that the combination of fenbendazole and levamisole resulted in efficacies of 62% when administered to goats with multiple resistant *H. contortus*. This reduction is not enough

to be clinically effective, but is more successful than the efficacy of 1 to 23% observed when the drugs were used solely (Miller and Craig, 1996).

An adequate strategy must be implemented to control resistant parasites in foals. Difficulty arises since one nematode is resistant to one class of drug, while another nematode in the same animal is resistant to another drug class. Drug combinations, as used in other hosts, may therefore be strategy to adequately control both parasites were resistance is found in foals.

CHAPTER III

MATERIALS AND METHODS

Management of horses

Twenty-eight Quarter Horse foals were utilized for the study. The study began when each foal was 60 d of age and ended between 174 and 270 d of age, when the youngest foal reached 174 d of age. All foals were from the breeding herd at Texas A&M University Horse Center and were managed consistently regarding vaccinations and health care. All foals were maintained at the Texas A&M University Horse Center, all experimental procedures were approved by the Institutional Agricultural Animal Care and Use Committee (AUP# 2007-68).

All foals were maintained on pasture from the time of birth until weaning at an average of 120 d of age. From d 120 until an average of d 200, they were housed as groups in dry lot pens, and fed individually twice daily. They were fed 1.5% of BW in concentrate and 0.75% of BW in Coastal Bermudagrass hay. At d 200, the foals were returned to pasture. All foals had ad libitum access to water. Each foal was weighed every 7 d before feeding or measured with a weight tape to allow for adjustment of their diets and accurate treatment dose.

Treatments

Foals were assigned into groups (n=4) based on their birthdates and then randomly assigned to a treatment regimen within their group (Table 1). Group I was first administered ivermectin (IVM) at 0.2mg/kg BW. Group II was first administered

ivermectin at 0.2 mg/kg BW and pyrantel pamoate at 6.6 mg/kg BW (IVM+PRT). Group III was first administered ivermectin at 0.2 mg/kg BW and fenbendazole at 10 mg/kg BW (IVM+FBZ). Group IV was first administered pyrantel pamoate at 6.6 mg/kg BW and fenbendazole at 10 mg/kg BW (PRT+FBZ). Each dosage was based upon the manufacturer's recommendation. Ivermectin was administered orally in a liquid (Merial Limited, Duluth, GA) and PRT (Columbia Laboratories, Lexington, KY) and FBZ (InterVet Inc., Millsboro, DE) in paste formulations. After 30 d, each group was administered a different drug/combination. That is group I was treated as group II, group II as group III, group III as group IV, and group IV as group I. Each group was rotated through the same treatment regimen and received each treatment at least once over a period of 4 mo.

Fecal analysis

Fecal samples were taken before treatment and 14 d post-treatment to determine the number of eggs (small strongyle and *Parascaris*) per gram of feces by confining foals to a 12'x12' stall and waiting until defecation occurred. Eggs per gram (EPG) were determined via the modified McMaster method (Herd, 1992) with a sensitivity of 50 eggs per g. Twenty-eight ml saturated salt solution (specific gravity of 1.20) and 2 g of feces were placed in a vial. The fecal material was well mixed and subsamples were removed from the mixture with a large bore pipette. The mixture was then added to a counting slide with two grids and examined under 100 x magnification. Eggs were counted and the total number of eggs in both grids was multiplied by 50 to achieve EPG

Table 1. Anthelmintic treatment regimen for foals (60 – 150 d)

Treatment groups	Drugs administered	Age of foals (days)
Group I	IVM 0.2 mg/kg	60
	IVM + PRT	90
	IVM + FBZ	120
	PRT + FBZ	150
Group II	IVM 0.2 mg/kg + PRT 6.6 mg/kg	60
	IVM + FBZ	90
	PRT + FBZ	120
	IVM	150
Group III	IVM 0.2 mg/kg + FBZ 10 mg/kg	60
	PRT + FBZ	90
	IVM	120
	IVM + PRT	150
Group IV	PRT 6.6 mg/kg + FBZ 10 mg/kg	60
	IVM	90
	IVM + PRT	120
	IVM + FBZ	150

(Herd, 1992). If *Parascaris* eggs were not detected by the McMaster method, then a 5-g Wisconsin double centrifugation (Todd et al., 1975) was performed, with a sensitivity of 0.2 EPG of feces. Five g of feces was mixed in tap water, and then strained using cheese cloth, and sedimented in water by centrifugation for 5 min at 275 *g*. The separated fluid was then removed. The remaining material, including eggs, was mixed into a sucrose solution (specific gravity of 1.27) and then spun up against a coverslip by centrifuging 10 min at 275 *g*. The coverslip was then placed on a slide and examined under 100x magnification and all eggs were counted to achieve eggs per gram (Todd et al., 1975).

Fecal egg count reduction (FECR) was calculated by the equation: [(Mean EPG prior to treatment – mean EPG 14 d post-treatment)/mean EPG prior to treatment] x 100.

Statistical analyses

Following the completion of fecal collections and determination of EPG in the fecal samples, the mean effects for each treatment were measured using the MIXED procedure of SAS (SAS Inst. Inc., Cary, NC) for repeated measures (Littell et al., 1996). The model contained effects for treatment and allowed for random effects of each individual. Main effects considered significant when $P < 0.05$ and considered a trend when $P < 0.15$.

CHAPTER IV

RESULTS

Small strongyles

Results of small strongyle FEC, for every treatment (n = 139 fecal samples) are presented in Table 2. Ivermectin was the only treatment that caused a significant ($P < 0.001$) decrease in FEC 14 d post treatment. Percent FEC reduction was also the highest at 99.72 ± 9.77%. There was a significant difference between treatments when comparing IVM vs. IVM+PRT ($P = 0.0018$), IVM vs. IVM+FBZ ($P = 0.0010$), and IVM vs. PRT+FBZ ($P < 0.0001$). There was a tendency for treatment with PRT+FBZ to be different from IVM+FBZ and IVM+PRT ($P < 0.11$) and no difference when comparing treatment with IVM+PRT vs. IVM+FBZ ($P = 0.83$). There was an increase of over 200% in FEC after treatment with PRT+FBZ, while all other treatments resulted in clinically significant reductions in FEC (Table 2).

Table 2. Fecal egg counts (eggs per g feces) for small strongyles found in foals-all treatments

Treatment	Before Treatment	14-d post treatment	Difference[a]	% Reduction[b]
IVM	29.47 ± 53.61	0.08 ± 0.27	29.39 ± 53.65[c]	99.72 ± 9.77
IVM+PRT	5.78 ± 18.44	0.34 ± 1.51	5.44 ± 18.18[d]	94.14 ± 69.70
IVM+FBZ	4.41 ± 16.93	0.57 ± 1.91	3.85 ± 17.16[d]	87.18 ± 133.97
PRT+FBZ	3.58 ± 9.32	11.91 ± 21.55	-8.32 ± 20.95[d]	-232.40 ± 626.65

[a] Difference obtained from Before treatment – 14 d post treatment

[b] % Reduction obtained from [(before treatment – 14 d post treatment)/before treatment] x 100

[c,d] Values in same column with different superscripts are different ($P < 0.05$)

Analysis was conducted using the first treatment data for each foal (n = 28 fecal samples). This was conducted since all treatments were given in the same order, and thus necessary to be able to distinguish effects from previous treatments. Results are presented in Table 3. The treatment of PRT+FBZ was significantly less effective than IVM+PRT ($P < 0.05$) and tended to be less effective than IVM ($P = 0.14$) and IVM+FBZ ($P = 0.12$). Again, treatment with PRT+FBZ resulted in an increase in FEC 14 d post treatment. The most successful treatment was IVM+PRT with a % reduction of 99.05%. Treatments with IVM and IVM+FBZ resulted in a positive reduction, but were below the expected efficacy (Table 3).

Table 3. Fecal egg counts (eggs per g feces) for small strongyles found in foals-first treatment

Treatment	Before Treatment	14-d Post Treatment	Difference	% Reduction
IVM	1.17 ± 1.39	0.31 ± 0.54	0.86 ± 1.49[c]	73.17 ± 20.37
IVM+PRT	9.00 ± 17.30	0.09 ± 0.15	8.91 ± 106.77[a]	99.05 ± 78.68
IVM+FBZ	2.94 ± 4.44	1.00 ± 2.39	1.94 ± 4.44[c]	66.02 ± 237.48
PRT+FBZ	3.88 ± 8.31	15.66 ± 40.26	-11.78 ± 8.31[b]	-303.78 ± 275.79

[a,b,c] Values in same column with different superscripts are different ($P < 0.05$)

Parascaris equorum

Results of *Parascaris* FEC for all treatments (n = 139 fecal samples) are presented in Table 4. There was no influence of treatment on *Parascaris* EPG ($P > 0.12$). However, when considering the % reduction, there was an increase in FEC post treatment when treated with IVM. Treatment with IVM+FBZ was the only treatment that resulted in a positive reduction that indicates the combination was effective. Treatments of IVM+PRT and PRT+FBZ resulted in positive FEC reductions, however, were below expected efficacies (Table 4).

Table 4. Fecal eggs counts (eggs per g feces) of *Parascaris equorum* eggs found in foals-all treatments

Treatment	Before Treatment	14-d post treatment	Difference	% Reduction
IVM	0.08 ± 0.30	2.10 ± 7.66	-2.02 ± 54.54[a]	-2646.15 ± 528.17
IVM+PRT	26.43 ± 75.70	12.71 ± 58.32	13.72 ± 98.03[a]	51.91 ± 81.96
IVM+FBZ	8.58 ± 28.92	0.11 ± 0.47	8.46 ± 28.85[a]	98.67 ± 1.45
PRT+FBZ	0.39 ± 1.22	0.27 ± 0.98	0.13 ± 1.11[a]	32.31 ± 19.89

[a] Values with same superscript are not significantly different ($P > 0.21$)

CHAPTER V

GENERAL DISCUSSION

In regards to small strongyles, the results of this study agree with those of previous studies. Both pyrantel and fenbendazole administration resulted in less than a 90% reduction in small strongyle egg counts 2 wk after treatment, which indicates resistance to these drugs (Drudge et al., 1979; Chapman et al., 1996; Lyons et al., 1996, Woods et al., 1998; Lyons et al., 1999; Lyons et al., 2001; Tarigo-Martinie et al., 2001; Kaplan, 2002; Kaplan et al., 2004). It is logical that a combination of these drugs (PRT+FBZ) would be less effective (difference of -8.32 EPG; $P < 0.05$) in controlling small strongyles in foals compared to the other treatments (IVM, IVM+PRT, IVM+FBZ) used in this study. Treatment with IVM resulted in the highest % reduction (99.72%), while PRT+FBZ caused an increase in FEC 14 after treatment.

Negative EPG differences or negative % reductions indicate a failure of the drug to control worm populations as a result of maturation and resistance. Ivermectin, when used alone or in combination, continues to be effective in controlling the small strongyle population in this study. However, the use of ivermectin combinations did not have any greater success than ivermectin used alone ($P < 0.002$). This could have been due to competitive inhibition of fenbendazole or pyrantel, which small strongyles are resistant to, with ivermectin, which they are not. More research must be conducted to confirm this finding.

Analyses based solely on the first treatment were conducted due to the fact that the prepatent period of small strongyles is 40 to 100 d (Reinemeyer, 1986; Lyons et al., 2000). All treatments were administered 30 d apart, so a subsequent treatment could have been administered before mature, egg producing adult worms were established in the large intestine. Since all treatments were administered in the same order with no wash out period in between, one treatment may have had an influence on the findings of a subsequent treatment. When a treatment is administered before an adult population is established, it would cause lower pre-treatment FEC, creating the appearance that a treatment was ineffective in controlling the population due to subsequent worm maturation when a greater post-treatment EPG was found. In addition, when a drug is administered, it only kills the nematodes residing within the lumen of the intestine. In the case of small strongyles, the encysted larval stages are protected from the action of the anthelmintics. In fact, it has been suggested that therapeutic removal of adult cyathostomes from the lumen may cause emergence of larvae from the intestinal wall (Reinemeyer, 1986). This is evident by the resumption of egg production from previously encysted strongyles soon after treatment.

The differences in EPG before and after treatment for *Parascaris* were not significantly different depending on treatment, but there was a tendency for ivermectin to be less effective in controlling the worm population. This is supported by a study conducted at the same facility (Craig et al., 2007) where ivermectin failed to reduce the *Parascaris* population in the horse. An increase in post-treatment EPG was observed, indicating maturation of ascarids already in the intestinal tract. A negative % reduction

(-2646%) was observed, indicating failure of ivermectin against *Parascaris*. Studies conducted elsewhere (Boersema et al., 2002; Hearn and Peregrine, 2003; Slocombe et al., 2007; Lyons et al., 2006; Lindgren et al., 2008) indicate the idea that ivermectin is not always effective in controlling *Parascaris* infections in young horses. The combinations of IVM+FBZ caused the highest % reduction (98.67%), while IVM+PRT and PRT+FBZ caused much lower, 51.91 and 32.31% respectively, and therefore are not considered clinically effective.

Again, perceived negative differences could have been due to worm maturation or indicative of resistance. However, since foals were treated consecutively with different treatments, if there had been an overwhelming success of a prior treatment before the ascarids reached maturity, it could have masked the perceived effects of a subsequent treatment. First treatment data for *Parascaris* was excluded from analysis due to the lack of positive before treatment fecal egg counts. This was probably due to the immaturity of the nematodes in foals at 60 d.

Failure to see significant results in this study could be due to the fact that the foals were first treated before worms reached maturity. Foals become infected soon after birth but the prepatent period, the time from infection until eggs are seen (indicating mature adult parasites), is 72 to 110 d (Clayton, 1986). Since females are extremely fecund and capable of laying greater than 50 million eggs per d per foal, care was taken to administer an anthelmintic before this time. Given that *Parascaris* eggs are extremely environmentally resistant (Clayton, 1986), it is advantageous to try and limit the number of eggs contaminating the pasture where the foals are housed to limit

infection of future generations of foals. Also, since *Parascaris* do not have the arrested development of larvae in the intestinal wall like cyathostomes, immature worms, maturing in the intestine for 2 mo prior to egg-laying capabilities, larvae should have been removed via anthelmintics before they reached maturity.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Since anthelmintic resistance is increasing among nematode populations that plague horses, an adequate control strategy must be put into place. Conclusions from this study were that no anthelmintic was more effective than others in controlling small strongyles and ascarids. However, it was found that the mixture of pyrantel and fenbendazole was ineffective in controlling small strongyle populations. Further research is needed to find an adequate strategy to control multiple nematodes in young horses that are resistant to different classes of anthelmintics.

If one were to conduct a similar study, it would be advantageous to make some changes. First, it would be better to wait until there was a positive diagnosis of adult worms of both species before administering the first treatment. Also be beneficial to increase the time period between treatments to allow reestablishment of parasites in the intestines to better test the efficacy of a subsequent anthelmintic. Finally, treatments should be randomized to as to get more accurate representation of the efficacy of a single drug/combination.

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APPENDIX

FECAL EGG COUNT RECORDS

ID	sequence	Str_before (EPG)	Par_before (EPG)	Treatment	Str_after (EPG)	Par_after (EPG)
1	1	0	0	I	0	0.6
2	1	0.2	0	I	0	0
3	1	3.8	0	I	1.2	0
4	1	0.6	0	I	0	0
5	1	2.2	0	I	1	0
6	1	0.2	0	I	0	0
7	1	1.2	0	I	0	0.2
23	2	5.4	0	I	0	42.8
24	2	2.8	0	I	0	0
25	2	1.6	0	I	0	0
26	2	47	0	I	0	0
27	2	55.4	0.2	I	0.2	0
28	2	1.2	0	I	0	0
17	3	0	0	I	0	0
18	3	11	0	I	0	0
19	3	100	0	I	0	0
20	3	21.4	0	I	0.2	0
21	3	17	0	I	0	9.2
22	3	100	0	I	0	0
11	4	5	0	I	0.2	0
12	4	22.2	0	I	0	0
13	4	150	0	I	0	0
14	4	250	0	I	0	0
15	4	3.6	0.8	I	0	8.4
1	5	35	0	I	0	0
2	5	2.4	0	I	0	0
3	5	0.4	0	I	0	0
4	5	2.4	0	I	0	0
5	5	100	1.6	I	0	10
6	5	10	0	I	0	0.2
7	5	32	0	I	0	0
23	6	16.6	0	I	0	0
24	6	1.4	0	I	0	0
25	6	0	0	I	0	0
8	1	0	0	I P	0	0

ID	sequence	Str_before (EPG)	Par_before (EPG)	Treatment	Str_after (EPG)	Par_after (EPG)
9	1	0.2	0	I P	0	0
10	1	0.2	0	I P	0.2	0
11	1	0.2	0	I P	0.4	0
12	1	9	0	I P	0	0
13	1	50	0	I P	0	350
14	1	0.2	0	I P	0	0
15	1	3.2	0	I P	0	0
1	2	0	300	I P	0	0
2	2	0	0	I P	0	16.2
3	2	0.6	18.4	I P	0	0
4	2	0	0	I P	0	0
5	2	0	0	I P	0	0
6	2	0	0	I P	0	0
7	2	0.6	250	I P	0	0
23	3	0	250	I P	0	1.4
24	3	0	0	I P	0	0
25	3	0.4	0	I P	0	0
26	3	0	0	I P	0	0
27	3	3.4	0	I P	8.8	0.8
28	3	1.4	0	I P	0.2	0.2
17	4	1.8	0	I P	0	0
18	4	100	13.4	I P	2.4	31.8
19	4	3	0	I P	0	0
20	4	8.4	0	I P	0.2	0
21	4	0.8	12.8	I P	0	0
22	4	20	0	I P	0	0
11	5	0	0	I P	0	0
12	5	2.2	0	I P	0	3.8
13	5	1.8	0	I P	0	3.8
14	5	0	100	I P	0	29.4
1	6	0.4	0	I P	0	13.2
2	6	0	0	I P	0	4.8
3	6	0.2	7	I P	0	2.2
4	6	0	0	I P	0	0
23	7	0	0	I P	0	0
16	1	0	0	I F	0	0

ID	sequence	Str_before (EPG)	Par_before (EPG)	Treatment	Str_after (EPG)	Par_after (EPG)
17	1	0	0	I F	0.2	0
18	1	2.4	0	I F	0	0
19	1	1.2	0	I F	6.4	0
20	1	4.2	0	I F	0.4	0
21	1	0.4	0	I F	0	0
22	1	12.4	0	I F	0	0
8	2	1	0	I F	2.2	0
9	2	0	0	I F	0	0
10	2	0	6.6	I F	0.2	0
11	2	0	0	I F	0	0
12	2	0	0.2	I F	0	0
13	2	0.2	75	I F	0	0
14	2	0	0	I F	0	0
15	2	0.2	0	I F	0	0
1	3	7.4	0	I F	0	0
2	3	0	0	I F	0	0
3	3	0.2	0	I F	0.4	0
4	3	0	0	I F	0	0
5	3	0.2	0	I F	0	0
6	3	6.8	0	I F	0	0
7	3	0	0	I F	0	0
23	4	0.2	6.6	I F	0	0
24	4	100	150	I F	0	0
25	4	0	0	I F	0	0
26	4	11.4	0	I F	0.2	2
27	4	0.2	0	I F	0	0
28	4	3.2	0	I F	0	0
17	5	0	0	I F	0	0
19	5	0.8	0	I F	0	0
20	5	0	0	I F	9.4	0
21	5	2	0	I F	0.2	0
11	6	0	0	I F	0	0
1	7	0	52	I F	0.2	2
2	7	0	9.8	I F	0	0
23	1	0	0	P F	0	0
24	1	3.2	0	P F	0.2	0
25	1	0.2	0	P F	0	0

ID	sequence	Str_before (EPG)	Par_before (EPG)	Treatment	Str_after (EPG)	Par_after (EPG)
26	1	20.8	0	P F	100	0
27	1	0	0	P F	8.4	0
28	1	0	0.2	P F	0	0
16	2	0	0	P F	25.2	0.8
17	2	0	0	P F	0	0
18	2	9	0	P F	8.8	0
19	2	0.8	0	P F	11.5	0
20	2	0	0.2	P F	0	0
21	2	0.2	0	P F	0.8	0
22	2	1.2	0.2	P F	10.4	0
10	3	0	0	P F	2.6	0
11	3	2	0	P F	0.2	0
12	3	0	0	P F	8.2	0
13	3	0	0	P F	55.2	0
14	3	0	0.2	P F	16	0
15	3	0.2	0	P F	1.8	0
1	4	3.2	0	P F	30.8	0
2	4	0.2	0	P F	0	0
3	4	0	0	P F	0	0
4	4	0	0	P F	0.4	0
5	4	3.2	0	P F	50	0
6	4	0	1.4	P F	37.2	0
7	4	0.4	0	P F	7.2	4.4
23	5	46.4	0	P F	15.6	0
24	5	0	0	P F	0	0
25	5	0	2.4	P F	0	0
26	5	2.2	0	P F	0.6	0
17	6	0	0	P F	0	0
18	6	22	2	P F	1.4	0
19	6	3	6.4	P F	0.4	3.6

VITA

Ashley Elizabeth Volker received her Bachelor of Science degree in animal science from The University of Vermont in 2006. She started her graduate career in August of 2006 at Texas A&M University-College Station, under the direction of Dr. Martha Vogelsang in the area of Equine Reproduction. While there she served as a teaching assistant for the Department of Biology. Her research interests include reproduction related to nutrition and the management of young horses.

Ashley Elizabeth Volker may be reached through the Department of Animal Science, Texas A&M University, 2471 TAMU, College Station, TX 77843-2471. Her email address is avolker@tamu.edu.